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Acute Decrease in HDL Cholesterol Associated With Exposure to Welding Fumes

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Abstract

Objective—To investigate acute changes in circulating lipids after exposure to relatively high levels of particulate matter through welding.

Methods—Using a repeated measures panel study, lipid levels before and after welding and personal exposures to fine particulate matter ($PM_{2.5}$) were measured in 36 male welders over 63 exposure and/or control days.

Results—There was a trend toward decrease in HDL (−2.3 mg/dL, P = 0.08) 18 hours after welding. This effect became significant (−2.6 mg/dL, P = 0.05) after adjustment for possible confounders. The effect was strongest (−4.3 mg/dL, P = 0.02) among welders who did not weld the day before the study. There were no significant changes in other lipids associated with welding or $PM_{2.5}$ exposure.

Conclusion—Welding exposure was associated with an acute decrease in circulating HDL, which may relate to the inflammatory and proatherosclerotic effects of fine particle exposure.

In the welding process, metal parts are heated to melting temperatures and joined together, releasing fumes of metal-rich fine particles that may be inhaled by welders and nearby workers. Studies have linked occupational welding with respiratory diseases, including pneumonia, lung cancer, and the development of asthma and chronic bronchitis.[1–4] Studies have also shown that welders are at higher risk for cardiovascular mortality and, specifically, ischemic heart disease.[4–7] These observations are consistent with the large body of evidence linking air pollution exposure and cardiovascular mortality and morbidity.[8–10] Cardiovascular mortality has been found to increase within hours of higher ambient particulate matter (PM) levels in cities, which suggests an acute intravascular reaction to the exposure.[11] While the precise mechanisms for these associations remain under investigation, there is evidence that particulate matter exposure in the lung may precipitate an intravascular inflammatory reaction that may, both acutely and chronically, contribute to atherosclerosis and ischemic heart disease.[8,9,12,13]

Several studies have linked particulate matter exposure with atherosclerosis. Cellular studies have found that $PM_{2.5}$ (fine particles measuring <2.5 μm), and particularly the smallest,

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ultrafine particles (measuring <0.1 μm) have a proinflammatory effect on cells by generating reactive oxygen species.[12,14] $PM_{2.5}$ travel deeply into the lungs and labeled synthetic particles of this size have been found to penetrate tissues and enter circulation rapidly after inhalation.[15,16] Previously, we reported associations between measured $PM_{2.5}$ exposure during welding and inflammatory cells and markers, including C reactive protein (a predictor of coronary heart disease).[17] A study of chronic exposure to ambient $PM_{2.5}$ in apolipoprotein deficient mice on a high-fat diet found a significantly larger mean aortic atherosclerotic plaque area, aortic lipid content, and macrophage infiltration in exposed mice.[18] A study of wild type rabbits found an increase in lipid pools in coronary and aortic plaques attributable to particulate matter exposure.[19] Similarly, a study in humans found a correlation between increases in $PM_{2.5}$ levels and carotid intimal-media thickness.[20] Araujo et al[13] found larger early atherosclerotic lesions, lower HDL antiinflammatory activity, and upregulation of antioxidant genes in genetically susceptible (apolipoprotein E deficient) mice chronically exposed to ultrafine air pollution.

We hypothesized that the atherogenic effect of fine particulate matter that leads to increased incidence of cardiovascular mortality may be mediated by acute changes in pro- and antiinflammatory circulating lipids. To investigate this, we measured circulating fasting lipid levels before and 18 hours after exposure to fine particulate matter generated through welding.

MATERIALS AND METHODS

Study Population

The study was approved by the Institutional Review Board of the Harvard School of Public Health (Boston, MA, USA). Written informed consent was obtained from each participant. The study population consisted of 36 boilermakers. Using a repeated measures panel study, participants were recruited and monitored at an apprentice welding school over four sampling periods: January/February 2004, February 2005, June 2005, and January/February 2006. In 2004, subjects were invited to participate as either welders or controls, whereas in 2006, participants who welded were invited to participate on a second weekend as controls. Participants who had participated in previous years also had the opportunity to participate again on subsequent study weekends. Of the 36 unique participants, 19 participated in more than one sampling period as a control or welder (13 participated twice, four participated three times and two participated in all four sampling periods). In all, 63 multiple measurements were taken in sets of pre- and postwelding blood samples. Forty-seven of these were collected before and after exposure to metal fume and airborne fine particulate matter through electric manual metal arc welding and metal inert gas welding on mild and stainless steel. The majority of the welding was performed on mild steel. Sixteen sets were from participants who served as controls and did not weld. Identical testing was performed on the controls, who spent the study period reading and watching movies in the union hall proper and were exposed primarily to background levels of particulate matter in the union hall. Of the 63 observation sets, 14 were collected from individuals who had welded the day prior to the study onset. A modified American Thoracic Society standardized respiratory questionnaire was used to collect information on medical history, smoking history, and occupational history of the participants.

The study took place in the union hall training facility of the Boilermakers Local 29 in Quincy, MA. Participants arrived on Saturday morning around 7:30 am at which time preshift health monitoring was performed. On welding days, the participants welded for about 6 hours, including a 30-minute lunch break. Participants returned the following morning for repeat health monitoring.

Exposure Assessment

Personal particle samplers were placed in the breathing zone of participants on their lapels, outside of the helmet, during their work or control day to monitor exposure to the particles with an aerodynamic mass median diameter <2.5 μm ($PM_{2.5}$). The KTL cyclone (GK2.05SH, BGI Incorporated, Waltham, MA, USA) with a 50% aerodynamic diameter cutpoint of 2.5 μm was used in line with a Vortex Timer 2 personal sampling pump (Casella USA, Amherst, NH, USA) calibrated at a flow rate of 3.5 l/min. The air sample was collected on a 37 mm polytetrafluoroethylene membrane filter (Gelman Laboratories, Ann Arbor, MI, USA) encased in a cassette and placed downstream of the cyclone. The filters were weighed before and after sampling on a MT5 microbalance from Mettler-Toledo Incorporated (Columbus, OH, USA) after equilibrating for a minimum of 24 hours in a temperature and humidity controlled room. The mass collected on the filter was divided by the air volume sampled to calculate the gravimetric $PM_{2.5}$ concentration.

Lipid Measurements

Fasting venous blood samples were collected on both mornings of the study period (preshift and the next morning) and were analyzed for fasting cholesterol, low-density lipoprotein, high-density lipoprotein, and triglycerides. Participants reported the time of their last meal at the time of blood collection. Samples were considered to be fasting if the reported time of the last meal or beverage was more than 9 hours prior to sample collection. In the few cases where the participant did not report the time of his last meal, nonfasting status was presumed. Of 126 total baseline and next morning blood draws, 96 samples were reported as fasting, 24 samples were nonfasting and eight did not identify the time of the last meal and were presumed to be nonfasting.

Statistical Analysis

Statistical analyses were performed using SAS version 9.1 (SAS Institute Incorporated, Cary, NC, USA). Exposure was investigated as both a dichotomized variable (nonexposed controls and welders) as well as a continuous variable using $PM_{2.5}$ concentration. The mean (standard deviation, SD) and median values of the $PM_{2.5}$ concentrations were determined for controls and welders. A two-sample *t* test was performed to compare the $PM_{2.5}$ concentrations in controls and welders.

Baseline and next morning mean (standard deviation) levels of total cholesterol, LDL, HDL, and triglycerides were determined by exposure status. To account for repeated measurements, linear mixed effects models were used to investigate the effect of welding fume exposure and $PM_{2.5}$ concentration on next morning lipid levels after adjusting for baseline levels. In a repeated measures study design, each participant serves as his own control by providing baseline lipid levels. This design has the advantage of inherently controlling for factors that may be predictive of lipid levels such as exercise, alcohol consumption, and family history of hyperlipidemia. In the adjusted model, data was additionally adjusted for age, statin use, fasting, smoking status, and welding the previous day. A similar model was used to investigate the linear exposure-response association between $PM_{2.5}$ concentrations as a continuous variable and lipid levels. Cook's Distance <0.5 was used as the criterion for assessing outlying residuals and influential data points. The level of significance for all analyses was set at $\alpha = 0.05$.

RESULTS

Study Population Characteristics

The study population demographic data are presented in Table 1. The study population included 36 men, of whom 34 served at least once as a welder, 14 served at least once as a

welder and later as a non-welding control, and two served once as a control only. Upon first entry into the study, 34 participated as welders and two as controls. Participant ages ranged from 24 to 63 years, with a mean age of 41 years (SD 11). Most participants (29 of 36) reported white race. Participants had a median of 5 years of boilermaker experience, ranging from 0 to 40 years. Twelve participants were current smokers, 10 were ex-smokers, and 14 had never smoked. In general, the study population consisted of relatively young, healthy workers, with few having underlying respiratory or cardiopulmonary diseases. Nine participants reported a history of asthma, hypertension, heart disease and/or diabetes (one reported both heart disease and hypertension, the others reported only one condition). Three participants were taking a cholesterol-lowering medication in the HMG-CoA reductase inhibitor (statin) class.

Particle Exposure Assessment

Particle samples were collected from all controls and welders during the workday. Personal particulate matter samples were collected for a mean sampling time of 6.1 hours (SD 1.5). Measured $PM_{2.5}$ concentration ranged from 0.01 mg/m^3 to 2.97 mg/m^3. The median $PM_{2.5}$ concentration was 0.05 mg/m^3 in controls and 0.65 mg/m^3 in welders. As expected, the $PM_{2.5}$ exposure of welders was significantly higher than controls (P = <0.001).

The composition of the $PM_{2.5}$ welding fumes has been reported in a previous study of this cohort during the same study period.[21] Using x-ray fluorescence to analyze the elemental content of the $PM_{2.5}$, the highest exposures were to iron, manganese, copper, zinc, sodium, potassium, silica, calcium, and sulfur.

Changes in Lipid Levels Following Welding Fume Exposure

The mean effects (95% CI) of welding fume exposure on lipid levels the following morning are shown in Table 2. In the crude model, there was a trend (P = 0.08) toward decrease in HDL levels of 2.3 mg/dL associated with welding, after adjusting for baseline HDL (95% Confidence Interval [CI]: −4.9 to 0.3). This effect was strengthened to a 2.6 mg/dL significant decrease (95% CI: −5.3 to 0.0, P = 0.05) after additionally adjusting for age, fasting status, use of a statin, current smoking, and welding the previous day. Restricting the analysis to fasting samples resulted in a similar effect size on HDL but with a wider confidence interval. Only baseline HDL levels and statin use were additional predictors of next day HDL (P < 0.001) in the multivariable model. There were no significant changes in the other lipid levels associated with welding in the crude or adjusted model. Some of the subjects (n = 14) performed welding the day before participating in the study. In these subjects, baseline lipid measurements may have been affected by the prior day's welding exposure and would be equivalent to an 18-hour postwelding lipid measurement. To account for this, we also performed a separate analysis on the 48 sets of blood samples from those who did not weld the day prior (one welder did not specify previous day welding status and was assumed to have welded the day before). The subgroup that welded the day before study onset was too small for a statistically meaningful subgroup analysis. In the subgroup (n = 48) who did not weld the previous day, there was a significant decrease in next morning HDL of 4.3 mg/dL (95% CI: −8.0 to −0.7, P = 0.02) after adjusting for age, fasting status, use of a statin, and current smoking. The results of the subgroup analysis are shown in Table 3.

The association between $PM_{2.5}$ concentrations and lipid levels was also examined. There were no statistically significant effects of $PM_{2.5}$ concentrations on any of the lipid levels (P ≥ 0.10).

DISCUSSION

In this study, welding was associated with a decrease in HDL levels 18 hours after welding exposure. This effect became greater in magnitude after adjusting for age, statin use, smoking, and fasting status. Although the effect size was small, it was statistically significant ($P = 0.05$) despite a relatively small study size (36 unique participants, 63 observation days) and the effect became larger and more significant ($P = 0.02$) in the subgroup analysis (48 observation days) among those who did not weld the day before the study. The trends for the other lipids did not approach statistical significance, suggesting that there may be a unique, acute effect of welding exposure on HDL only.

Although the measured $PM_{2.5}$ levels were significantly greater among welders compared to controls ($P < 0.0001$), no exposure–response relationships were found between $PM_{2.5}$ exposure and any lipid levels. One possible explanation for the lack of an association between HDL levels and $PM_{2.5}$ exposure despite the association found with welding is that the level and range of $PM_{2.5}$ exposure was too small to detect an exposure–response relationship. Compared to our earlier study, at this welding school in 2003 and 2004 in which significant exposure–response relationships were detected between $PM_{2.5}$ exposure and several inflammatory markers, including C reactive protein and white blood cell counts,[17] the $PM_{2.5}$ exposure of welders at the school has decreased substantially. The median $PM_{2.5}$ exposure in our previous study had a median concentration of 1.69 mg/m^3 among welders, compared to 0.65 mg/m^3 in the present study.[17] Our mean and median $PM_{2.5}$ concentrations among welders were less than the 25th percentile exposure level seen in the study by Kim et al,[17] likely reflecting improved working conditions at the welding school. With a lower and narrower distribution of $PM_{2.5}$ exposure levels, the probability of finding a statistically significant effect was likely reduced. The $PM_{2.5}$ samples were placed outside of the face shield, which could lead to some exposure misclassification. It is also possible that a different exposure related to welding other than fine particulate matter was the cause for the decreased HDL level, such as stress, noise, or level of activity.

There may be mechanistic plausibility for an effect on HDL associated with elevated levels of PM through welding activity. The findings of this study and others that have observed effects of fine and ultrafine particulate matter exposure on lipids could shed light on the link between air pollution and atherosclerosis. Atherosclerosis is increasingly thought of as a complex inflammatory process in which lipids play a central role. LDL particles, once oxidized, activate the endothelial cells to produce adhesion molecules and chemotactic proteins that attract monocytes to the vessel wall and induce differentiation to macrophages in an inflammatory reaction.[22] Monocyte-derived macrophages internalize LDL after it is oxidized, forming foam cells.[23] Increased levels of LDL oxidation has been found to be a sensitive marker for coronary artery disease and predictive of cardiovascular risk.[24] It is believed that particulate matter has a proinflammatory effect on cells by generating reactive oxygen species.[12] There is evidence that transition metals or other oxidants on the particle surface may cause LDL oxidation directly and increase oxidation of LDL by monocytes, which may mediate the proatherogenic effect of PM exposure.[25]

HDL, on the other hand, is known to protect against cardiovascular disease. Traditionally, this protective effect has been attributed to HDL's role as an acceptor of cholesterol in reverse cholesterol transport back to the liver. There is growing evidence that HDL may exert an antiinflammatory effect by reversing the recruitment of monocytes that is triggered by LDL oxidation.[26] HDL inhibits oxidation of LDL and perhaps also oxidative stress of macrophages through its associated enzymes paraoxonase and lecithin: cholesterol acyltransferase (LCAT).[26–28] Mertens et al[26] found accelerated atherosclerosis in obese mice associated with decreased HDL-associated enzyme activity and increased LDL

oxidation and macrophage infiltration. Samples of HDL from mice exposed to fine and ultrafine particulate matter have been found to have significantly reduced HDL antiinflammatory properties than unexposed mice, suggesting that PM exposure may reduce the ability of HDL to protect against atherosclerosis.[13] Specifically, when pooled plasma HDL of exposed mice was added to human arterial wall cultures with human LDL, there were higher levels of inflammatory cell migration to the arterial wall compared to unexposed mice and this effect was highly significant after exposure to both fine and ultrafine particles.[13] Interestingly, Araujo et al[13] found a statistically significant increase in total cholesterol levels in exposed mice, but no decrease in HDL levels. Takano et al[29] found that exposure to nitrogen dioxide, which has also been found to oxidize lipids,[30,31] was associated with decreased HDL levels in both obese and nonobese rats. Yeatts et al[32] examined ambient PM exposure and lipid levels in 12 asthmatics and found statistically significant positive associations between ambient course $PM_{2.5–10}$ levels and triglycerides and VLDL, but no associations with $PM_{2.5}$ exposure and HDL.[32] There are also some gene expression data supporting an effect of PM exposure on lipids and inflammation. Gong et al[22] found that ambient ultrafine PM chemicals increased in vivo expression of genes related to vascular inflammation and lipid metabolism.

Though a small number of recent studies have found effects on lipid levels, function and gene expression patterns associated with PM exposure, there is of yet no general consensus on what the exact mechanism may be. Our study found a decrease in circulating HDL levels 18 hours following exposure to elevated levels of PM through welding activity, but no exposure–response relationship with measured PM concentration. The effect of welding exposure on HDL was strengthened after correcting for welding the previous day, which is the expected result and supports the conclusion that previous day welding influences next morning HDL levels. Our study was not sufficiently powered to assess an exposure–response relationship between HDL and PM and no significant effect was found (though the trend was in the negative direction). It remains to be seen whether a transient change in circulating HDL levels is of clinical or mechanistic significance. As HDL is known to protect against atherosclerosis and is believed to have an antiinflammatory effect at the endothelial level, a decrease in HDL following PM exposure is consistent with the known association between PM exposure and cardiovascular risk. Further studies are needed to elucidate the acute effects of PM exposure on lipid metabolism and function. The authors hope that this study will inspire future research to elucidate the association between exposure to air pollution and cardiovascular morbidity and mortality.

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TABLE 1

Demographic Characteristics of Male Welders in the Study Population*

Age, years	
Mean (SD)	41 (11)
Median	39
Range	24–63
Race (n)	
White	29
Black	2
Hispanic	3
Other	2
Years as boilermaker	
Mean (SD)	10 (11)
Median	5
Range	0–40
Smoking status (n)	
Current	12
Ex-smoker	10
Never smoker	14
Chronic disease (n)	
Asthma	3
Hypertension	3
Heart disease	3
CHF†	0
Diabetes	1
Statin use (n)	3

*Data is for the 36 unique participants upon first entry to the study.

†CHF, congestive heart failure.

TABLE 2

Mean Effects of 6 to 8 Hours of Welding (Yes/No) on Next Morning Lipid Levels

	Crude Model			Adjusted Model*		
	β	**95% CI**	***P* value**	***β***	**95% CI**	***P* value**
Total Cholesterol, mg/dL	−4.0	(−11.3−3.4)	0.28	−1.0	(−7.7−5.8)	0.77
Triglycerides, mg/dL	11.3	(−21.8−44.4)	0.49	11.0	(−26.8−48.8)	0.55
HDL, mg/dL	−2.3	(−4.9−0.3)	0.08	−2.6	(−5.3−0.0)	0.05
LDL, mg/dL	−2.7	(−10.8−5.4)	0.50	−0.5	(−9.4−8.4)	0.90

*Models adjusted for baseline lipid level, age, fasting, use of statin, smoking, and welding the previous day.

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TABLE 3

Mean Effects of 6 to 8 Hours of Welding (Yes/No) on Next Morning Lipid Levels Among Those Who Did Not Weld the Previous Day

	Adjusted Model*		
	β	**95% CI**	***P* value**
Total Cholesterol, mg/dL	−2.6	(−10.8−5.6)	0.50
Triglycerides, mg/dL	9.3	(−44.0−62.6)	0.71
HDL, mg/dL	−4.3	(−8.0−0.7)	0.02
LDL, mg/dL	−0.6	(−12.8−11.7)	0.92

*Models adjusted for baseline lipid level, age, fasting, use of statin, and smoking.